

Hypercapnia-induced cerebral oedema in a patient with COPD exacerbation: a rare and under-recognised entity

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DESCRIPTION

A 56-year-old Caucasian woman with severe chronic obstructive pulmonary disease (COPD) on home oxygen presented with a severe exacerbation. Vitals included a blood pressure of 111/73 mm Hg, a pulse of 102/minute, respiratory rate of 13/minute and O₂ saturation of 84% on oxygen of 2–3 L/minute by nasal cannula. She was lethargic, confused and was wheezing. She was placed on bi-level positive airway pressure (BiPAP) but her mentation continued to deteriorate. White cell counts were normal. Chest X-ray showed hyperinflated lungs but no infiltrates. Arterial blood gases revealed severe hypercapnic respiratory acidosis with a pH of 7.26, pCO₂ of 100.1 mm Hg, pO₂ of 187 mm Hg and bicarbonate of 47 mmol/L. In spite of BiPAP, her acidosis worsened with a pCO₂ of 131 mm Hg. She was intubated for acute hypercapnic respiratory failure. Although her deteriorating mentation correlated with CO₂ narcosis, it was judicious to perform neuroimaging like cerebral CT to rule out other causes of acute mental decompensation, particularly cerebrovascular accidents. Given this context, a CT of the head without contrast was performed which was negative for haemorrhage or ischaemia but revealed effacement of sulci, hypodense white matter, and vastly decreased size of lateral ventricles and basal cisterns consistent with cerebral oedema

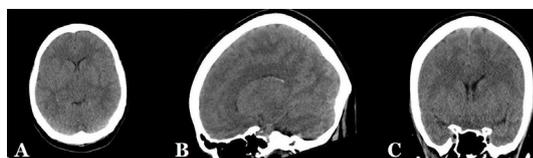


Figure 1 CT head without contrast (A) axial view (B) sagittal view (C) coronal view showing effacement of sulci, hypodense white matter, loss of grey-white matter junction differentiation, decreased size of lateral ventricles and basal cisterns consistent with cerebral oedema in the presence of hypercapnic respiratory failure.



Figure 2 CT head without contrast (A) axial view (B) sagittal view (C) coronal view showing a near-complete recovery of cerebral oedema after the resolution of hypercapnia and improvement of respiratory failure.

Learning points

- ▶ While cerebral perfusion varies optimally physiologically, an extreme variation in pathological states, like trauma, stroke, infection and hypercapnia, can lead to cerebral oedema.¹
- ▶ Patients with chronic obstructive pulmonary disease can have a dual mechanism of cerebral hyperperfusion including hypercapnia and hypoxia which can lead to cerebral oedema with features suggestive of raised intracranial pressure (nausea, vomiting, drowsiness, confusion, pupillary dilation, papilloedema, stupor and coma) in addition to changes in respiratory status.^{1,2}
- ▶ Imaging including CT, MRI, positron emission tomography and transcranial Doppler can help in cerebral perfusion assessment and can exclude other causes of altered mentation like haemorrhage or ischaemia.² Treatment in such cases requires correction of the underlying pathology and resolution of hypercapnia, sometimes requiring intubation.³
- ▶ Although, hypercapnia-induced cerebral hyperperfusion is well known, hypercapnia-induced cerebral oedema remains an under-recognised entity and hence should be considered in the absence of other causes of raised intracranial pressure.
- ▶ The treatment of this entity should focus on the underlying pathophysiology and hence on hypercapnia as the main cause of hyperperfusion and cerebral oedema.

(figure 1). She received usual treatment for COPD exacerbation including steroids and antibiotics. The following day her pCO₂ was 78 mm Hg with corresponding improvement in her cerebral oedema on CT (figure 2). Mental status continued to improve and she was extubated after 2 days.

Contributors SC is the primary author who had initially written the preliminary rough draft of the image and then did primary data gathering; the accuracy of the data was checked and confirmed with the intensivist. AW collected information upon imaging and description of the neuroimaging and probed the findings further. He also rewrote the case and gave it the final shape. SV did the literature search and revised the whole article. SJS checked the language aspect of the case and made sure that the information is accurate and understandable. She gave the final shape to the article.

Competing interests None declared.



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